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=> s p53 and review/dt
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L1          4534 P53 AND REVIEW/DT

=> s l1 and py<1997
L2          1340 L1 AND PY<1997

=> s l2 and p53/ti
L3           273 L2 AND P53/TI

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L4   ANSWER 1 OF 273     MEDLINE on STN
AN   96306066     MEDLINE
DN   PubMed ID: 8763583
TI   [p53 antibodies: a new method for the analysis of alterations of
     the p53 gene: application to breast cancer].
     Les anticorps anti-p53: une nouvelle methode d'analyse des
     alterations du gene p53: application au cancer du sein.
AU   Soussi T; Peyrat J P; Lubin R; Bonneterre J
SO   Pathologie-biologie, (1996 Apr) 44 (4) 232-4.  Ref: 13
     Journal code: 0265365. ISSN: 0369-8114.
CY   France
DT   Editorial
       General Review; (REVIEW)
     (REVIEW, TUTORIAL)
LA   French
FS   Priority Journals
EM   199610
ED   Entered STN: 19961219
     Last Updated on STN: 19980206
     Entered Medline: 19961028
AB   Alterations in the p53 gene are found in 20% to 40% of breast
     cancers and are generally associated with factors of adverse prognostic
     significance.  In most instances, point mutations modify the confirmation
     of p53, causing the gene to accumulate in the nuclei of tumor
     cells.  These alterations can be detected via molecular analysis or
     immunohistochemical methods.  More recent studies have demonstrated that
     accumulation of the p53 protein in tumor cells may induce an
     immune response with presence of anti-p53 antibodies in the
     serum of cancer patients.  Assaying serum anti-p53 antibody is a
     new approach to investigation of the status of the p53 gene in a
     tumor.

L4   ANSWER 2 OF 273     MEDLINE on STN
AN   97000048     MEDLINE
DN   PubMed ID: 8843191
TI   Strange bedfellows in even stranger places: the role of ATM in meiotic
     cells, lymphocytes, tumors, and its functional links to p53.
CM   Comment on: Genes Dev. 1996 Oct 1;10(19):2401-10. PubMed ID: 8843193
     Comment on: Genes Dev. 1996 Oct 1;10(19):2411-22. PubMed ID: 8843194
     Comment on: Genes Dev. 1996 Oct 1;10(19):2423-37. PubMed ID: 8843195
AU   Hawley R S; Friend S H
CS   Department of Genetics, University of California at Davis, 95616, USA.
SO   Genes & development, (1996 Oct 1) 10 (19) 2383-8.  Ref: 49
     Journal code: 8711660. ISSN: 0890-9369.
CY   United States
DT   Commentary
     Journal; Article; (JOURNAL ARTICLE)
       General Review; (REVIEW)
     (REVIEW, TUTORIAL)
LA   English
FS   Priority Journals
EM   199611
ED   Entered STN: 19961219
     Last Updated on STN: 19961219
     Entered Medline: 19961127
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L4 ANSWER 3 OF 273 MEDLINE on STN
AN 97194830 MEDLINE
DN PubMed ID: 9042268
TI The dual role of mutant **p53** protein in chemosensitivity of human cancers.
AU Mueller H; Eppenberger U
CS Department of Gynecology, University Hospital Basel, Switzerland.
SO Anticancer research, **(1996 Nov-Dec)** 16 (6B) 3845-8. Ref: 25
Journal code: 8102988. ISSN: 0250-7005.
CY Greece
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199703
ED Entered STN: 19970407
Last Updated on STN: 19970407
Entered Medline: 19970327
AB Mutational loss of **p53** tumor suppressor functions has been observed in a wide range of neoplasms and was associated with either enhanced or decreased chemosensitivity of affected tumors. The dual role of wild-type **p53** as a DNA repair initiator and a trigger for apoptosis raises the possibility that appropriately designed chemotherapy could be selectively applied against **p53**-defective tumor cells. The cytotoxic effects of DNA-crosslinking chemotherapeutica such as cisplatin could be enhanced by mutated **p53** which is no longer able to repair drug-induced DNA damage. In contrast, DNA synthesis blockers such as fluorouracil can induce apoptosis through **p53** -dependent mechanisms. Thus, loss of **p53** functions results in decreased sensitivity to this type of drugs. Clinical studies will reveal the role of abberant **p53** in the efficacy of chemotherapy for individual patients.

L4 ANSWER 4 OF 273 MEDLINE on STN
AN 96344733 MEDLINE
DN PubMed ID: 8741682
TI Role of the **p53** gene in apoptosis.
AU Takahashi R; Shinohara H
CS Department of Pathology and Tumor Biology, Graduate School of Medicine, Kyoto University.
SO Nippon rinsho. Japanese journal of clinical medicine, **(1996 Jul)** 54 (7) 1881-7. Ref: 31
Journal code: 0420546. ISSN: 0047-1852.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA Japanese
FS Priority Journals
EM 199611
ED Entered STN: 19961219
Last Updated on STN: 19961219
Entered Medline: 19961126
AB Cell numbers are controlled by a homeostatic mechanism between cell growth, arrest and programmed cell death (apoptosis) in normal and cancerous tissues. One of the tumor suppressor genes, **p53**, functions as a transcription factor or transcriptional regulator through DNA and protein binding properties, and plays an important role in regulating cell cycle and induction of apoptosis. Although there are two apoptotic pathways, **p53**-independent and **p53**-dependent, the latter will be emphasized and discussed in this section. Since **p53** is often inactivated due to mutation in human cancers, understanding the **p53**-dependent apoptotic pathway is extremely important. Analysis of **p53**-dependent apoptosis as well as apoptosis caused by other **p53**-related genes should provide a clue to a new strategy for cancer therapy.

L4 ANSWER 5 OF 273 MEDLINE on STN
AN 96438659 MEDLINE
DN PubMed ID: 8841019
TI Structure and function of the **p53** tumor suppressor gene: clues for rational cancer therapeutic strategies.
AU Harris C C
CS Laboratory of Human Carcinogenesis, Division of Basic Science, National Cancer Institute, Bethesda, MD 20892-4255, USA.
SO Journal of the National Cancer Institute, **(1996 Oct 16)** 88 (20) 1442-55. Ref: 288
Journal code: 7503089. ISSN: 0027-8874.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199611
ED Entered STN: 19961219
Last Updated on STN: 19961219
Entered Medline: 19961106
AB The **p53** tumor suppressor protein is involved in multiple central cellular processes, including transcription, DNA repair, genomic stability, senescence, cell cycle control, and apoptosis. **p53** is functionally inactivated by structural mutations, interaction with viral products, and endogenous cellular mechanisms in the majority of human cancers. This functional inactivation can, in some circumstances, produce resistance to DNA-damaging agents commonly used in cancer chemotherapy and radiotherapeutic approaches. Current research is defining the biochemical pathways through which **p53** induces cell cycle arrest and apoptosis. Knowledge of these fundamental processes is leading to the identification of molecular targets toward which multimodality cancer therapies, using chemotherapeutic, immunotherapeutic, and gene-therapeutic strategies, can be based.

L4 ANSWER 6 OF 273 MEDLINE on STN
AN 96203992 MEDLINE
DN PubMed ID: 8622853
TI New insights into **p53** function from structural studies.
AU Arrowsmith C H; Morin P
CS Division of Molecular and Structural Biology, Ontario Cancer Institute, University of Toronto, Canada.
SO Oncogene, **(1996 Apr 4)** 12 (7) 1379-85. Ref: 59
Journal code: 8711562. ISSN: 0950-9232.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199606
ED Entered STN: 19960627
Last Updated on STN: 19960627
Entered Medline: 19960618
AB Recent structural analysis of **p53** has greatly enhanced our understanding of the biochemical activities of this protein by presenting us with a detailed picture of the chemical groups in the protein that are involved in protein stability, conformation and functional interactions. The current structures form the basis for the design of potential therapeutics which could, for example, revert a DNA-binding mutant back to a DNA-binding competent conformation. The structure of the tet domain forms the basis for designing an active therapeutic **p53** with an oligomerization domain which would not cross react with a DNA-binding mutant **p53**. However, as useful as these structures have been in providing insight into the structure/function relationship for **p53** , a complete understanding of this protein awaits more detailed information on the full-length protein. In this respect, one of the most useful roles for future structural studies will be to help identify the nature of the conformational transition between latent and active

p53, and how it can be modulated.

L4 ANSWER 7 OF 273 MEDLINE on STN
AN 96288984 MEDLINE
DN PubMed ID: 8710022
TI [Hereditary mutations in the **p53** tumor suppressor gene; significance for clinical practice. National Work Group Hereditary Mamma Carcinoma].
Erfelijke mutaties in het **p53**-tumorsuppressorgen; betekenis voor de klinische praktijk. Landelijke Werkgroep Erfelijk Mammacarcinoom.
AU Menko F H; Nooy M A; Vasen H F
CS Academisch Ziekenhuis Vrije Universiteit, afd. Klinische Genetica, Amsterdam.
SO Nederlands tijdschrift voor geneeskunde, **(1996 Jun 29)** 140 (26) 1347-50. Ref: 21
Journal code: 0400770. ISSN: 0028-2162.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA Dutch
FS Priority Journals
EM 199609
ED Entered STN: 19960919
Last Updated on STN: 19960919
Entered Medline: 19960912

L4 ANSWER 8 OF 273 MEDLINE on STN
AN 97069847 MEDLINE
DN PubMed ID: 8912827
TI Lymphoepithelial carcinoma of the larynx and hypopharynx: study of eight cases with relationship to Epstein-Barr virus and **p53** gene alterations, and review of the literature.
AU MacMillan C; Kapadia S B; Finkelstein S D; Nalesnik M A; Barnes L
CS Department of Pathology, University of Pittsburgh Medical Center, PA, USA.
SO Human pathology, **(1996 Nov)** 27 (11) 1172-9. Ref: 44
Journal code: 9421547. ISSN: 0046-8177.
CY United States
DT (CASE REPORTS)
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW OF REPORTED CASES)
LA English
FS Priority Journals
EM 199701
ED Entered STN: 19970128
Last Updated on STN: 19970128
Entered Medline: 19970107
AB Eight cases of lymphoepithelial carcinoma (LEC) of the larynx and hypopharynx were evaluated for clinicopathologic features, and the presence of the Epstein-Barr virus (EBV) and **p53** alterations. The seven men and one woman, all of non-Asian descent, averaged 64 years of age. Eighty-eight percent had histologically confirmed cervical lymph node metastasis at diagnosis. None had systemic disease. Seven of eight patients available for follow-up (mean, 17.7 months) were alive and free of disease, although one did develop recurrent tumor in the neck. Four tumors were composed, histologically, of pure LEC. Four others had foci of both LEC and conventional squamous cell carcinoma. All eight tumors exhibited alterations in **p53** expression, but none was positive for EBV. Combining these 8 cases with the 15 previously published cases in the English literature indicate that LEC in this site is a rare, rather aggressive tumor, primarily of older adults (mean, 62 years) with a propensity for early cervical lymph node metastasis and eventual distant dissemination and death from disease in about one third of patients. Although **p53** alterations are common and of no apparent prognostic significance, LEC at this site seems to have little, if any, relationship to the EBV in patients of non-Asian origin.

L4 ANSWER 9 OF 273 MEDLINE on STN

AN 96206040 MEDLINE
DN PubMed ID: 8654922
TI **p53**: puzzle and paradigm.
AU Ko L J; Prives C
CS Department of Biological Sciences, Columbia University, New York, New York 10027, USA.
NC CA58316 (NCI)
SO Genes & development, **(1996 May 1)** 10 (9) 1054-72. Ref: 245
Journal code: 8711660. ISSN: 0890-9369.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA English
FS Priority Journals
EM 199607
ED Entered STN: 19960808
Last Updated on STN: 19970203
Entered Medline: 19960726

L4 ANSWER 10 OF 273 MEDLINE on STN
AN 96247678 MEDLINE
DN PubMed ID: 8644842
TI The two faces of tumor suppressor **p53**.
AU Smith M L; Fornace A J Jr
CS Laboratory of Molecular Pharmacology, Developmental Therapeutics Program, National Cancer Institute, Bethesda, Maryland 20892, USA.
SO American journal of pathology, **(1996 Apr)** 148 (4) 1019-22. Ref: 39
Journal code: 0370502. ISSN: 0002-9440.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199607
ED Entered STN: 19960726
Last Updated on STN: 19960726
Entered Medline: 19960712